

CONDITIONED INHIBITION IN FLAVOR AVERSION LEARNING:
ODOR AS A CONDITIONED INHIBITOR

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CONDITIONED INHIBITION IN FLAVOR AVERSION LEARNING:

ODOR AS A CONDITIONED INHIBITOR

by



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ABSTRACT

Two experiments were performed which demonstrated that an olfactory stimulus can become associated with toxicosis over long CS-US delays and, more importantly, that an olfactory stimulus can become a conditioned inhibitor in a feeding situation. In Experiment 1, hooded rats were allowed to drink water while a stream of amyl acetate vapor was directed towards the end of the drinking spout. Toxicosis was then induced via the injection of lithium chloride after delays of 0, 0.5, 1, 4, and 12 hrs for different groups. When compared with no-toxicosis controls, it was found that significant aversions were obtained for all groups except the 12 hr delay group. In Experiment 2, hooded rats were given conditioned inhibition training in which the taste of saccharin alone was always followed by induced illness, but the taste of saccharin plus the odor of amyl acetate was not. In a series of three subsequent tests -- summation, enhancement of conditioning, and retardation -- it was demonstrated that the odor had acquired active inhibitory properties. The results paralleled those obtained with more traditionally studied stimuli and techniques and hence were found to be readily predictable from a recent model of conditioning set forth by Rescorla & Wagner (1972).

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INTRODUCTION

Animals which experience illness following ingestion of a flavored substance will tend to form an aversion to that substance (Revusky & Garcia, 1970; Rozin & Kalat, 1971). Such a flavor-toxicosis association develops even when a period of several hours intervenes between the two events (Revusky, 1968; Smith & Roll, 1967). This ability of rats to associate flavor with illness over long delays contradicts the principle that temporal contiguity between the CS and US is necessary for learning. Rozin & Kalat (1971) have interpreted this apparent difference between flavor-toxicosis association and traditionally studied types of learning as evidence that flavor aversion learning is a different, more primitive form of learning that has evolved as a specialized adaptation for the control of feeding behavior. They have claimed that complex, "cognitive" types of learning do not readily occur in flavor aversion paradigms (Kalat & Rozin, 1972). By "cognitive" they apparently mean that an animal's behavior reflects the influence of complex stimulus interactions. In contrast, Revusky (1971) has insisted that the basic learning processes are the same in all types of learning, and that differences between learned flavor aversions and other types of learning are entirely due to a principle called "stimulus relevance" (Capretta, 1961). Flavors readily become associated with toxicosis and do not readily become associated with other USes; furthermore, sickness readily becomes associated with flavor stimuli and not with other types of CSes. Hence, consumption of a flavored substance

can be separated from toxicosis by a long delay and conditioning. can still occur because the events occurring during the delay do not substantially interfere with the flavor-toxicosis association. Revusky (1971) explicitly denies any other basic difference between flavor aversion learning and other types of learning.

Revusky's position has been supported, first of all, by evidence that learned associations over long delays can occur in traditional learning paradigms if events occurring during the delay are prevented from interfering with the referent association (Lett, 1973; Lett, in press; Pschirrer, 1972; Revusky, 1973). Secondly, evidence has accumulated which has suggested that flavor aversion learning is not the primitive form that Rozin and Kalat supposed it to be. Recent experiments (Lavin, 1973; Revusky, 1971) have demonstrated that Kamin's (1969) blocking effect, Pavlov's (1927) overshadowing effect, and the sensory-preconditioning effect are obtainable from within flavor aversion paradigms; all of these effects could be considered quite "cognitive."

Another complex, rather esoteric type of learning is conditioned inhibition. Here, a stimulus comes to control a tendency opposite to that of a conditioned excitor as a result of a conditioning procedure in which the excitor signals the occurrence of a US, and the presence of the inhibitor signals the absence of that same US (Rescorla, 1969). In traditional paradigms, it has been shown that, once an animal has assimilated this information, it can use it in a seemingly insightful manner. The present series of experiments attempted to show that this effect is obtainable in the feeding situation, and that it exhibits

the same "cognitive properties" which it has manifested under traditional conditions. Such a demonstration would argue strongly in favor of Revusky's position, while casting additional doubt on the belief held by Rozin and Kalat that flavor aversion learning is somehow "primitive."

Rescorla (1969) has deemed two principle tests necessary for the proper demonstration of conditioned inhibition. These are:

a) summation, in which the simultaneous presentation of a CS- (inhibitor) with a CS+ (excitor) results in a weaker response than when the CS+ alone is presented, and b) retardation, in which the conditioning of an excitatory response is slower to a CS- than to a novel stimulus. Although these two methods are in themselves deemed sufficient to identify a stimulus as a conditioned inhibitor, Rescorla (1971a), has presented, as an implication of a theory (Rescorla & Wagner, 1972), a test which can be used to provide even further evidence of inhibition. In this procedure, which shall be called enhancement of conditioning, a CS- is presented in compound with a neutral stimulus, CS_n , and this compound is then reinforced. If CS- is truly an inhibitor, than conditioning to CS_n will be increased when compared with appropriate controls.

In flavor aversion learning, the typical excitatory response to a conditioned stimulus is a reduction in consumption of the food with which the stimulus is related. A conditioned inhibitor must then be a food-related stimulus which actively counters this response. Some indication as already been provided by Best & Hubbard (1973) that flavor stimuli may acquire such inhibitory properties. A flavor

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solution which was explicitly negatively correlated with illness in rats was found to be preferred over a third solution or water. However, this increase in preference is not strong evidence that conditioned inhibition in flavor aversion situations has the same properties as conditioned inhibition in the traditional situation; it was not demonstrated that the alleged inhibitor possessed any of the active/inhibitory properties which Rescorla (1969) has claimed for conditioned inhibition. A reason for this may be that Rescorla's tests pose methodological difficulties for flavor aversion experiments. First of all, stimulus compounding is called for. Flavor stimuli, however, cannot be presented simultaneously in compound unless they are mixed together, and this necessitates concern over potential masking effects. Secondly, it is required that the animal re-experience already aversive stimuli. Flavor stimuli are normally experienced as a result of a voluntary feeding response by the animal, unless some sort of elaborate technique (Domjan & Wilson, 1972; Bradley & Mistretta, 1971) is used. Therefore, for the demonstration of conditioned inhibition in the feeding situation to be practical, some modification of the manner in which the stimuli are presented in feeding experiments was necessary.

A solution to these problems was suggested by the finding that aversions may be created to odors as well as to tastes (Garcia & Koelling, 1967). Odors as stimuli are involuntarily experienced and may be presented simultaneously with tastes to form stimulus compounds. Their primary disadvantage is that it is difficult to confine them to a specific location and to limit their presentation to a given

duration; these difficulties may have been responsible for the prevalent supposition that odor aversions are weaker than taste aversions (Garcia & Koelling, 1967; Hankins, Garcia, & Rusiniak, 1973). However, in a preliminary experiment (to be presented herein as Experiment 1) it was demonstrated that, once an odor is adequately controlled, it may be used somewhat as readily as tastes by rats for the regulation of food intake. The necessary apparatus included a chamber designed for rapid air exchange and a specially devised drinking spout which simultaneously delivered both unflavored water and a stream of odorized air to the rat. Toxicosis was induced after the rats consumed the water in the presence of the odor. In subsequent tests, presence of the odor decreased the amount of water consumed, indicating an aversion to the odor. A strong odor aversion occurred after a single pairing with an odor-toxicosis delay of as much as four hours. This suggested that odor stimuli might readily be compounded with tastes in feeding experiments; to form such a compound, the unflavored water that was presented together with the odor need only be replaced with any flavored solution.

This method was then employed in a demonstration of conditioned inhibition within a flavor aversion paradigm (Experiment 2). Initially, the rats were given conditioned inhibition training to the odor of amy~~l~~ acetate. This was accomplished by always following a taste stimulus alone (ie., saccharin solution) with lithium chloride-induced toxicosis; on days in which the odor of amyl acetate was presented simultaneously with the saccharin solution, however,

toxicosis was never induced. When it became clear that the rats had learned this discrimination, they were then subjected to the three tests of conditioned inhibition in the following order: summation, enhancement of conditioning, and retardation. If the odor had acquired true inhibitory properties as a result of conditioning, then it was expected that, during these tests, it would exhibit properties identical to those which have been demonstrated for inhibitory stimuli of other modalities.

In the summation test (Test Phase I), it was expected that the simultaneous presentation of the inhibitory odor (CS-) with an aversive taste (CS+) other than the saccharin used during the inhibitory training would result in a weaker response than when the taste alone was presented. For the enhancement of conditioning test (Test Phase II), the stimulus compound of the inhibitory odor (CS-) plus a neutral taste (CS_n) was followed by illness. If the odor was truly an inhibitor, then it was expected that conditioning to the neutral taste would be increased when compared with appropriate controls. Finally, in the retardation test (Test Phase III), the rate of conditioning to the odor was expected to be slower for those animals which had received inhibitory training of the odor than for those which had not.

EXPERIMENT 1

When an animal has learned an aversion to a flavored substance, it has probably come to associate both the olfactory and gustatory components of a flavor stimulus with a toxic aftereffect. A learned aversion to an odor would serve to prevent the repeated ingestion of the toxic substance from which the odor emanates. Garcia & Koelling (1967) first demonstrated that rats can learn an aversion to water which has been odorized via the placement of an odorant near the drinking spout, the water remaining unadulterated by the odorant liquid itself. Although a significant aversion resulted, it was considerably weaker than a taste aversion. Later studies also indicated that odor can become an aversive stimulus when tested in a variety of ways, including response suppression (Lorden, Kenfield, & Braun, 1970), olfactory discrimination (Supak, Macrides, & Chorover, 1971), and reduction of water consumption (Domjan, 1973).

In all of the studies just mentioned, illness was experimentally induced immediately after an animal consumed a solution which was paired with an odor. In light of the success of these attempts, it was expected that odor-toxicosis associations could develop over long delays, as has been shown for tastes. However, a study by Hankins, Garcia, & Rusiniak (1973) seemed to indicate the contrary. They found that a delay of 30 min between an odor-water pairing and illness eliminated the aversion entirely, and a delay of 10 min weakened it substantially. Unfortunately, in contrast with the earlier odor experiments, they were unable to produce a strong

one-trial aversion even in the absence of a delay. Supak et al. (1971) already suggested that the difference in strength between the odor and taste aversions in the Garcia & Koelling (1967) study resulted from inadequate stimulus control. The same criticism might be levelled here. Hankins et al. (1973) made little attempt to spatially or temporally isolate the odor; instead, it was allowed to permeate the experimental room. Extrapolating from more traditional conditioning paradigms, it is probable that a discrete, localized CS is more readily associated with the US than is a diffuse one, since discrete stimuli have commonly been used with success in experiments involving other sensory modalities (Gormezano & Moore, 1969). Olfactory stimuli have typically been avoided because of difficulties involved in limiting their presentation.

The purpose of Experiment 1 was to demonstrate that aversions to odors can be created over long CS-US delays when care is taken to adequately control the olfactory stimulus. A chamber was constructed which was designed to allow a rapid and constant exchange of air. Also, an odor presentation system was devised which delivered a regulated stream of odorant vapor directly to the end of the drinking spout. These refinements together resulted in a high degree of localization of the odor to the liquid to be consumed, as well as a relatively accurate control of stimulus onset and offset.

Method

Subjects and Apparatus. Forty-eight Long-Evans hooded rats, weighing 300-350 g, were housed in individual wire cages and were given free access to Purina chow throughout the experiment. The

animals were deprived of water for two days prior to the experiment and were then limited to 10 min of water per day on all days including the recovery day. All drinking took place in the experimental apparatus.

The experimental chamber consisted of a plexiglass box capable of accomodating two of the wire cages in which the rats were housed. The box, measuring 30.5 x 61.0 x 30.5 cm, was sealed at all seams in order to make it as airtight as possible. Its front panel acted as a door, swinging outward and upward. When closed, this panel was drawn shut tightly against a Teflon seal mounted on the edges of the open end of the box. On the ceiling of the chamber, two runners were mounted from which two wire cages could be suspended. The cages were placed into the chamber from the front and the door panel closed flush against their front walls. Attached to the front of the door panel were two metal C-clips, each capable of holding a 300 ml bottle for one of the cages. Just below each C-clip, a 1.5 cm hole had been drilled to accomodate a water spout. Thus two bottles could be clipped to the door panel in such a way that their drinking spouts protruded through the panel and into the cages at approximately a 3.5 cm drinking level for the rats. At the top of the chamber, two 7.6 cm square holes were cut and screened with activated charcoal filters. A 10.2 cm exhaust fan situated at the rear of the chamber drew air out of the box and into 10.2 cm plastic tubing through which the air was carried to the outside of the building. This fan was in operation continuously throughout all experimental sessions.

The drinking spouts were designed to create the illusion that the odor was emanating from the liquid to be consumed. They were

made of glass and consisted, basically, of a tube within a tube. The inner glass tube transmitted the liquid from the bottle, and the surrounding tube transmitted purified or odorized air. Air was pumped into this spout by a Gelman air pump via 0.5 cm I.D. Silastic Teflon coated tubing. Prior to reaching the spout, the air passed through two activated charcoal filters and then through two 1000 ml gas-washing bottles containing either 100 ml of mineral oil (for trials in which only purified air was presented) or a combination of the odorant liquid plus mineral oil (for trials in which odorized air was presented). A three-way Teflon stopcock placed between the filters and the gas-washing bottles served as an on/off valve for the air flow.

Procedure. On Days 1-5, all animals were given 10 min of water in the experimental chamber per day. Two rats at a time were moved, each in its own cage, from the housing rack to the chamber, which was located in a separate room. The door panel was shut and sealed, and then a bottle containing room-temperature tap water was presented to each of the animals for 10 min. At the same time, a 1500 cc/min stream of pure air was pumped to them via the drinking tubes. During each session of this familiarization procedure as well as throughout the experiment, amount consumed was measured by weighing the water bottles before and after each 10 min drinking period.

On the treatment day (Day 6), the forty-eight animals were divided into six groups of eight animals each. On this day, the animals were allowed their usual 10 min of drinking in the chamber, but amyl acetate vapor rather than purified air was presented to

them. That is, the purified air was first passed through the two gas-washing bottles containing 10 ml of amyl acetate suspended in 90 ml of mineral oil. Following the drinking session, all rats except those in the no-toxicosis control group were injected with 2% body weight of 0.15 molar lithium chloride solution. For the different experimental groups, the injections occurred at different times after removal from the chamber: at 0, 0.5, 1, 4, and 12 hrs. The control animals received no injection but were simply returned to the housing rack following the session.

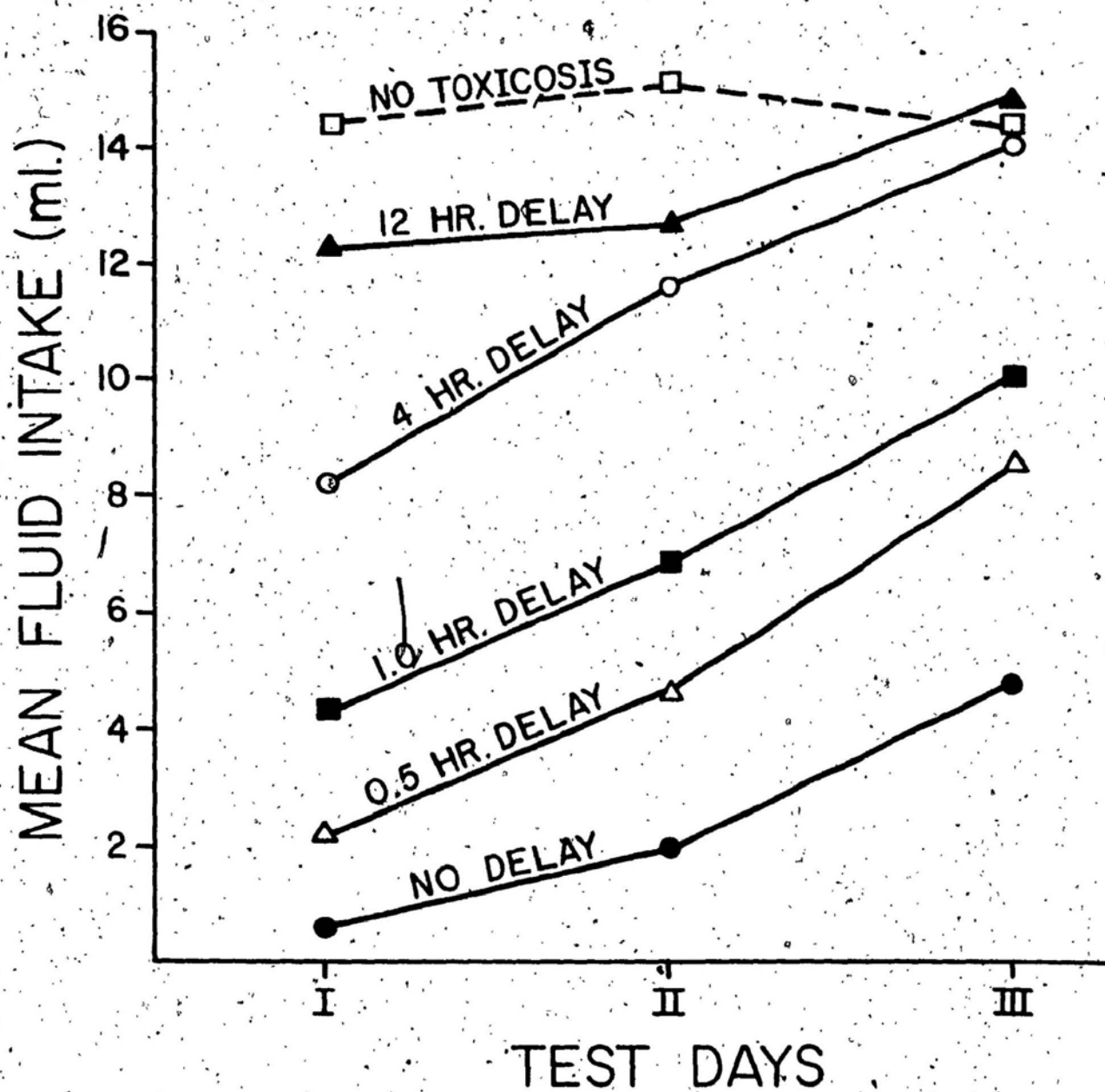
In cases in which a rat exhibited marked neophobia to the odor and would not drink, it was not injected but was replaced on the housing rack. Four hours later, it was again subjected to the odor-water pairing. All neophobic animals drank during this second trial and were injected with lithium after the appropriate delay.

One recovery day was allowed in which the rats were given 10 min of water in the presence of purified air while in the test apparatus. On the following day (Day 8), they were tested for aversions to the odor by presentation of water paired with amyl acetate vapor for 10 min. Similar additional tests occurred on Days 11 and 14. On the intervening days, the odor was absent during the drinking session.

Results and Discussion

Figure 1 shows an aversion to water paired with odor for all groups except the 12 hr delay group and the no-toxicosis control. An analysis of variance performed on the data from the first test day across all six groups revealed a significant treatment effect

Figure 1. Mean water consumption during the odor-water pairing sessions on the three test days.



($F = 39.45$, $df = 5/42$, $p < .001$). However, the 12 hr group did not differ significantly from the no-toxicosis group ($t = 1.49$, $df = 14$, $p > .05$), although a clear difference emerged between the 4 hr and 12 hr groups ($t = 3.05$, $df = 14$, $p < .01$). A repeated measures analysis on all trials for the groups which clearly showed an aversion (groups 0, 0.5, 1, and 4 hrs) yielded a significant effect as a result of difference in delay ($F = 12.78$, $df = 3/28$, $p < .001$) and as a result of extinction ($F = 65.88$, $df = 2/56$, $p < .001$). No discernible differences were found between the animals that had exhibited neophobia to the odor at its initial presentation and those that had not; intra-group t -tests between the two types of animals all yielded $p > .05$ (two-tailed).

These results demonstrated that odor-toxicosis associations can readily occur even over a delay of as much as four hours. The success of the present study can probably be attributed to the relatively high degree of control over the stimulus. It might be objected that the high degree of localization of the odor to the water caused the water to become tainted by odor molecules going into solution on the rat's tongue, so that this experiment did not result in true odor aversions. Although some degree of taste stimulation may very well have been produced, it seems unlikely that this alone could have accounted for the strong aversions obtained. This assumption can be made in light of evidence presented by Garcia (1971) and Dragoin (1971) that, when other factors remain constant, the strength of an aversion becomes a function of flavor intensity; that is, the weaker the concentration of a given flavor, the weaker

the ensuing aversion. Presumably, the taste produced by the odor molecules was a weak one at best; however, the aversions which resulted over all delays were quite comparable in magnitude to aversions produced to a .25% w/v saccharin solution over similar delays via basically similar techniques, including the injection of an identical dosage of lithium chloride (Nachman, 1970). This suggests that the odor itself served as the primary aversive CS.

In the Hankins et al. (1973) experiment, the olfactory system did not seem to adhere to the same principles of one-trial learning and long-delay reinforcement that are common to the gustatory system. It was pointed out that there exists neurological evidence which has suggested that the afferents of the olfactory system do not project directly to the nucleus solitarius of the brain stem as do the gustatory and visceral afferents, but rather terminate primarily in the limbic system. Consequently, the authors argued that olfaction must play a minor role in the regulation of feeding behavior, and that its primary function is to serve as a telereceptor. The present experiment, however, seemed to indicate the contrary. It was apparent from the results that olfactory cues can be used in much the same way as taste cues for the regulation of food intake.

EXPERIMENT 2

Experiment 1 demonstrated that an olfactory cue can acquire excitatory properties as a result of being paired with illness and, hence, that rats are capable of using odors to regulate their food intake. The purpose of Experiment 2 was to demonstrate that this same olfactory cue can also acquire inhibitory properties in the manner of more traditionally utilized stimuli, in spite of the fact that conditioning of this type would appear to be maladaptive in the natural feeding situation.

Inhibition Training

Rescorla (1969) has pointed out that the most common technique for producing conditioned inhibition involves the negative correlation of a CS with a US. In the present instance, the odor of amyl acetate (CS) was negatively correlated with toxicosis (US). Two groups of rats, Groups CI and CIC, were subjected to a training procedure in which consumption of saccharin solution in the absence of the odor of amyl acetate was paired with toxicosis induced by the injection of a lithium chloride solution, while consumption of saccharin solution in the presence of the odor was not followed by toxicosis. A third group, Group NCI, was subjected to a latent inhibition control procedure; it was treated exactly like the conditioned inhibition groups except that toxicosis was never induced. According to Rescorla (1971b) and Reiss & Wagner (1972), this latent inhibition procedure should not endow the odor with active inhibitory properties.

Method

Subjects. Three groups of eight male Long-Evans hooded rats, each weighing 275-300 g, were housed in individual wire cages and were given free access to Purina chow throughout. They had no access to fluid, however, except as part of the experiment. The animals were deprived of water for two days prior to training in order to hasten habituation to the apparatus and were then limited to 10 min of fluid a day on all days including recovery days. All drinking took place in the experimental apparatus.

Apparatus. The same olfactory apparatus as had been employed in Experiment 1 was used throughout all stages of Experiment 2.

General Procedure. The same basic procedure was followed on each day throughout inhibition training, as well as throughout the subsequent test phases. The rats were moved, two at a time, each in its own cage, from the housing rack to the experimental chamber. The door panel was shut and sealed, and then a bottle containing either unflavored water or a taste solution was presented to each of the animals for 10 min. At the same time, a 1500 cc/min stream of filtered air was pumped to them via the drinking spouts. On the appropriate occasions, the air was odorized with amyl acetate vapor. That is, prior to reaching the drinking spouts, the air passed through the two gas-washing bottles which each contained 10 ml of amyl acetate and 90 ml of mineral oil instead of the 100 ml of mineral oil alone. Fluid consumption was measured by weighing the bottles before and after each 10 min drinking session.

Conditioning. For the first seven days, the rats received 10 min per day of access to unflavored water in the absence of any odor in order to habituate them to the experimental apparatus and the deprivation procedure. Conditioning began on Day 8. On this day, all animals received a 0.5% w/v sodium saccharin solution instead of the unflavored water. Following removal from the chamber, animals in Groups CI and CIC were immediately injected intraperitoneally with 1% body weight of a 0.15 molar lithium chloride solution and then returned to the housing rack. Rats in group NCI received a placebo injection of 1% body weight of normal saline. One day was allowed for recovery. The procedure on all recovery days was the same as during pretraining: 10 min of water together with purified air while in the apparatus.

For the next ten days thereafter (Days 9-19), all groups were treated alike. The saccharin solution was again presented to all the animals and was paired with amyl acetate vapor. No injections were administered. When it was found that, by the third day of these compound stimulus presentations (Day 12), most of the animals that had received the lithium chloride were drinking none or very little of the saccharin solution, it became necessary to permit repeated trials on the same day. These trials were continued on a given day until each animal had consumed at least 5 ml. As a result of this procedure, all animals were drinking readily by Day 14.

On Day 20 a series of three-day cycles was begun. The first day's session consisted of the same procedure employed on Day 8: a saccharin-toxicosis pairing for groups CI and CIC and a saccharin-

saline pairing for Group NCI. The second day was allowed for recovery. On the third day, the saccharin solution was again presented together with amyl acetate vapor, and no injections were administered. This cycle was repeated four times, until the animals in Group CI and CIC drank virtually none of the saccharin solution at all on a day when the odor was not present.

Following the conclusion of the fourth cycle, three extra days of odor-saccharin pairings were given to the rats, until no significant difference in fluid consumption existed between the two groups having experienced illness (CI and CIC) and the placebo group (NCI).

Test Phase I

The summation test of conditioned inhibition requires that a supposed conditioned inhibitor (CS-) be presented simultaneously with a known excitator (CS+). If the response elicited by the CS- and the CS+ in compound is less than the response to the CS+ alone, then the CS- may be said to have acquired inhibitory properties. In flavor aversion learning, the CSes are odors and tastes, the US is an induced illness, and the response is typically the reduction in consumption of a substance ingested prior to the onset of illness. Inhibition of this response would therefore mean an increase in intake of the substance which was paired with toxicosis. In the present test phase, a sour taste (dilute hydrochloric acid) was followed by illness for all three groups of rats, thus conditioning this taste as an excitator. This CS+ was subsequently presented to each of the three groups either a) in compound with the odor of amyl acetate, where the odor was a conditioned inhibitor (Group CI), b) in compound with the odor of amyl acetate, where the odor was a latently inhibited stimulus (Group NCI), or c) alone, in the absence of the odor of amyl acetate, although the odor had originally undergone inhibition training (Group CIC). In other words, on the test days, Group CI received a combination of CS- and CS+; Group NCI received a combination of CS+ and a latently inhibited CS; and Group CIC received the CS+ alone. If the odor had truly acquired active inhibitory properties, then it was expected that the response to the aversive sour taste (CS+) should be least for Group CI; hence, animals

of this group should consume more of the sour solution than animals of either control group. Group CIC served as an indicator of the amount of excitatory conditioning that would normally occur to the sour taste alone; and Group NCI served to demonstrate that any attenuation of the aversion in Group CI was actually due to inhibition training, and not simply to increased familiarity with the odor.

Method

Subjects. All twenty-four animals which had undergone the previous inhibition training were used in this test phase and were maintained on 10 min a day of fluid.

Procedure. On day 35, a 1.5% v/v solution of 1.0 normal hydrochloric acid was presented to the rats during their 10 min drinking period in the apparatus. Immediately afterward, animals in all three groups were injected intraperitoneally with 1% body weight of a 0.15 molar lithium chloride solution and were then returned to the housing rack. The next day was allowed for recovery, with 10 min of unflavored water in the absence of the odor to be consumed in the apparatus. The summation test was administered on Days 37, 39, and 41. Rats in Groups CI and NCI were presented with the dilute HCl paired with amyl acetate vapor for 10 min in the apparatus; Group CIC received only the dilute HCl in the absence of any odor. Water only was presented on intervening days.

Results and Discussion

The acquisition of active inhibitory properties by the odor of amyl acetate was confirmed by means of the summation test. Figure 2 shows that Group CI, subjected during testing to an odor


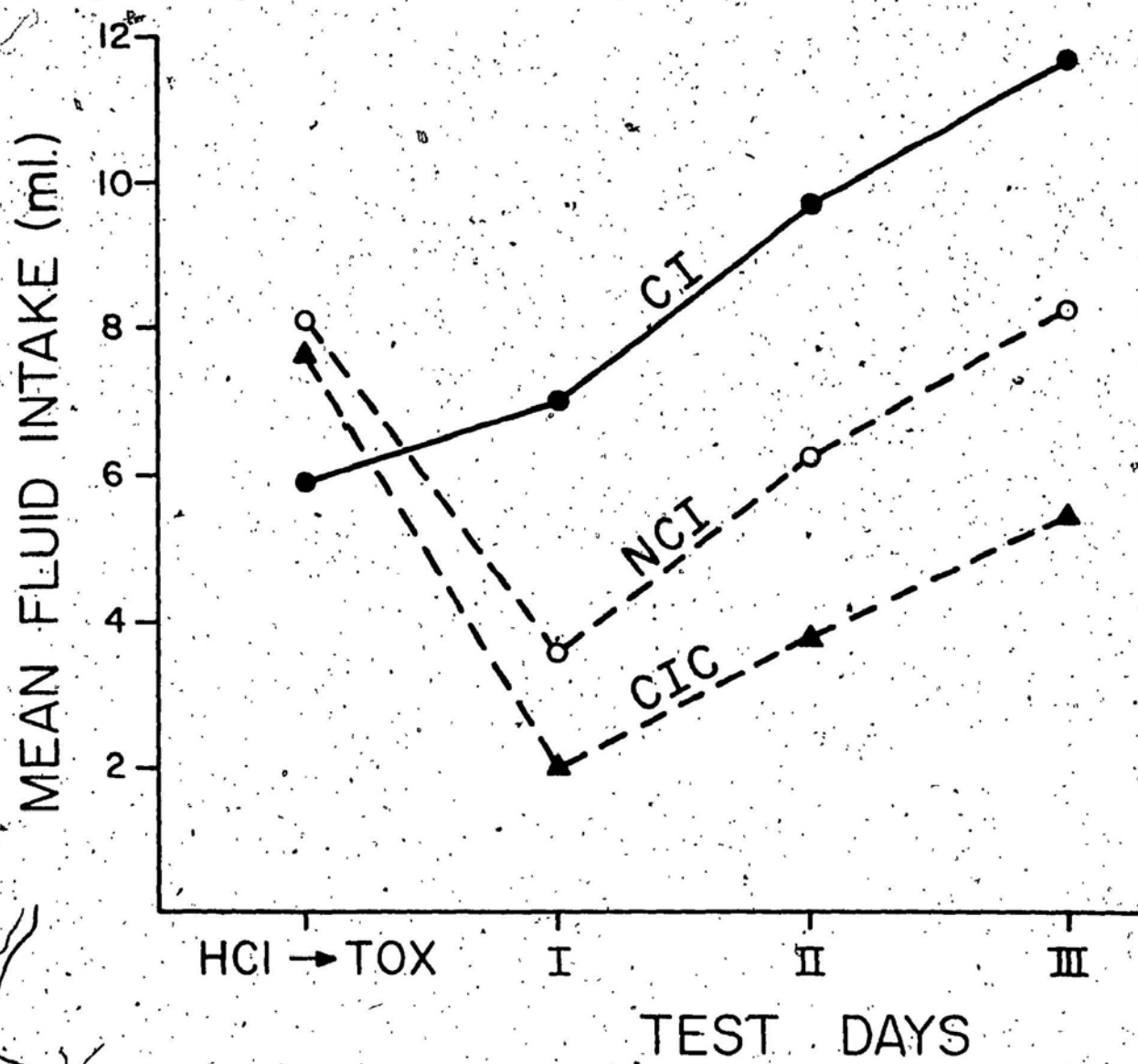


Figure 2. Mean intake of hydrochloric acid on the treatment and test days. On the treatment day, the HCl solution was presented alone and was followed by toxicosis; on the test days, the HCl solution was presented either in the presence of an inhibitor odor (Group CI), in the presence of a familiar odor (Group NCI), or in the absence of any odor (Group CIC).



which was a conditioned inhibitor, drank more of the aversive HCl solution than rats subjected to an equally familiar odor which was not a conditioned inhibitor (Group NCI) or rats which consumed the HCl solution in the absence of the odor stimulus (Group CIC). A repeated measures analysis of variance over the three test days yielded $p < .001$ ($F = 10.01$, $df = 2/21$) for differences between groups and $p < .001$ ($F = 53.66$, $df = 2/42$) for an extinction effect. Separate analyses of variance between pairs of groups confirmed the fact that Group CI had drunk more than either Group NCI ($F = 5.23$, $df = 1/14$, $p < .05$) or Group CIC ($F = 23.10$, $df = 1/14$, $p < .001$).

The difference between Groups NCI and CIC was not significant under a two-tailed test ($F = 3.79$, $df = 1/14$, $p > .05$) but would have been significant at the .05 level had it been hypothesized a priori on a one-tailed basis that the presence of the latently inhibited odor during testing should increase consumption of the aversive HCl solution. If this result were to prove reliable, it would seem at first glance to show that latent inhibition produces a weakened conditioned inhibition effect. However, the same result could be explained more parsimoniously in a different way. For Group NCI, there was a difference between training and test conditions because the odor was absent during HCl aversion training but present during the test. There was no such difference for Group CIC because the odor was absent during both training and test days. Since a change of conditions from training to testing generally attenuates conditioned effects, such a result does not contradict the finding by Rescorla (1971b) and Reiss & Wagner (1972) that latent inhibition lacks the active inhibitory properties ascribed to conditioned inhibition.

Test Phase II

It has been well established that if two CSes precede a single US, the positive association of one CS with the US tends to interfere with the association of the second CS with the US. For instance, if both a tone and a light are presented prior to shock, the more strongly the one CS becomes conditioned, the more it interferes with the conditioning of the other CS (Kamin, 1969; Rescorla & Wagner, 1972). Similarly, if two flavored substances are consumed prior to the same instance of toxicosis, the stronger the learned aversion to one substance, the greater the interference with the learning of the aversion to the second substance (Revusky, 1971). Rescorla & Wagner (1972) have recently presented a theory of Pavlovian conditioning which adequately describes this phenomenon and, as a logical progression, postulates the occurrence of its symmetrical counterpart. That is, if excitatory conditioning of a stimulus increases its ability to produce interference, then inhibitory conditioning should reduce this ability. This postulate is derived from the bipolar nature of excitation and inhibition: an excitatory CS predicts the occurrence of a US, and an inhibitory CS predicts the non-occurrence of a US. An implication of this theory is that, if, for example, a tone and a light are presented prior to shock, conditioning of the light ought to be stronger if the tone is a conditioned inhibitor than if it is equally familiar but is not a conditioned inhibitor. In other words, because the inhibitory tone has a low associative strength relative to shock, it interferes less with the light-shock

association than if it were merely familiar but not inhibitory. Rescorla (1971a) has confirmed this prediction in conditioned suppression experiments; it remains to be shown here that this effect, which we shall call enhancement of conditioning, can be obtained in a feeding experiment as well.

In Test Phase II, the odor of amyl acetate was paired with a novel taste (sodium chloride solution) for all rats, and this compound was then followed by induced illness. Preference for the salt solution was subsequently tested by presenting it alone in the absence of the odor. In light of the Rescorla (1971a) results, it was expected that the presence of the odor on the treatment day would result in less interference with the taste-illness association for the experimental group (CI), which had received inhibitory training of the odor, than for the control group (NCI) which had not experienced this training. That is, on the test days, Group CI was expected to show a greater aversion to the salt solution than Group NCI. Thus, the second source of evidence for conditioned inhibition would be provided. (Group CIC was discarded for this test phase, as well as for Test Phase III, because it would not have yielded any important information.)

The same animals of Groups CI and NCI of Test Phase I were used here. At first, it was expected that Test Phase II would serve only as a pilot study, since, as a result of the treatment and test days of Test Phase I, Group NCI had received some conditioned inhibition training; hence, it could no longer be considered a perfect control. However, it was found that this minor bit of

inhibitory training was by no means comparable in magnitude to that experienced by Group CI during the original inhibition training.

This became clear during the enhancement of conditioning test.

Thus the results proved to be quite convincing, even though only marginal results had been expected.

Method

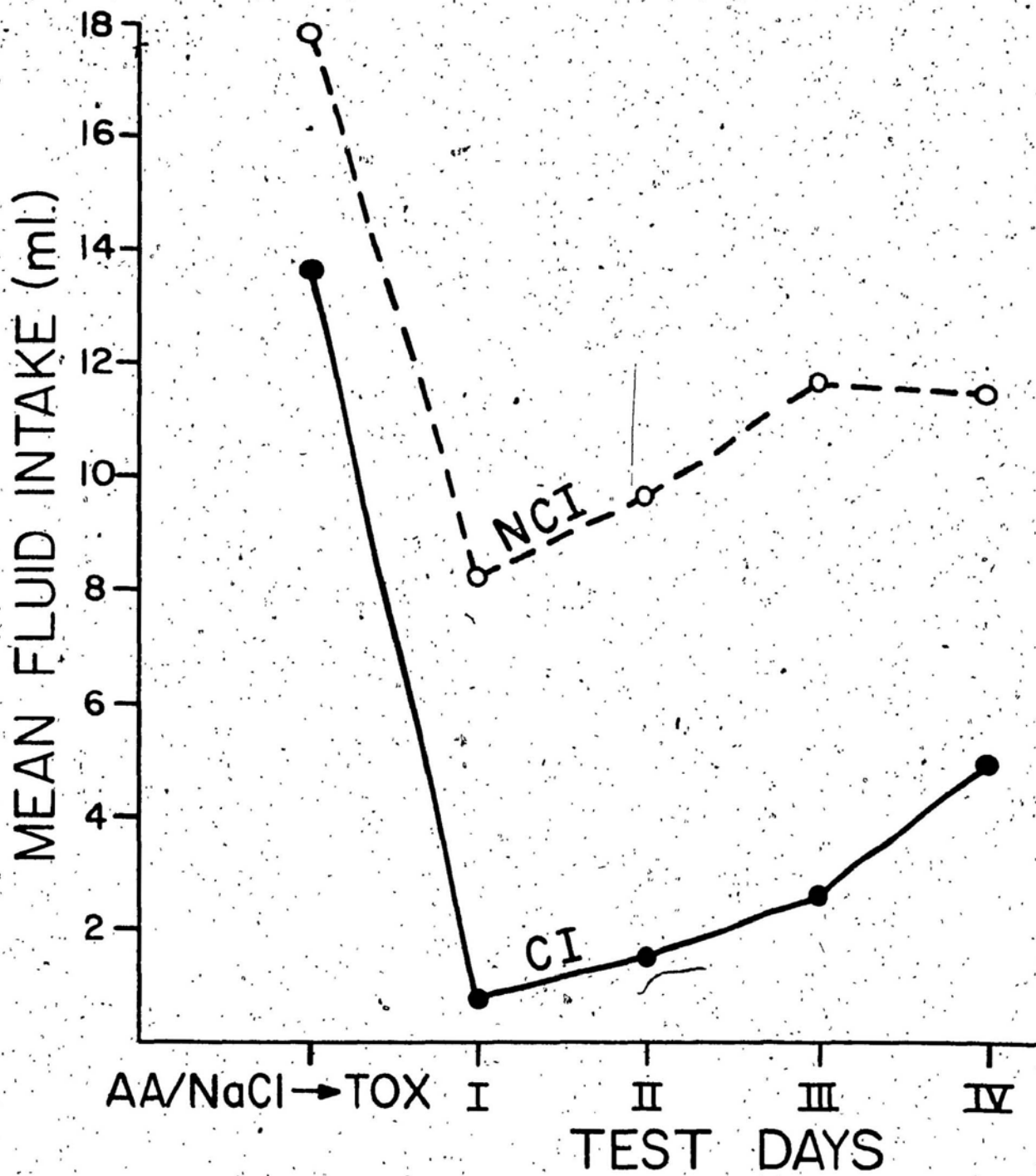
Subjects. The sixteen animals of Groups CI and NCI of the previous test phase were used, and these retained their group designations.

Procedure. On the day after the third test day of Test Phase I, water only was presented. On the following day (Day 43), all sixteen rats were presented with a 1.5% w/v sodium chloride solution together with amyl acetate vapor. This was followed immediately by an injection of 1% body weight of a 0.15 molar lithium chloride solution. One recovery day was allowed, followed by four test days (Days 45, 47, 49, and 51) on which only the sodium chloride solution was presented, in the absence of amyl acetate vapor. Water only was permitted on intervening days.

Results and Discussion

The results of this test showed that the previous inhibitory training of the odor of amyl acetate increased the amount of conditioning to the salt solution as a result of reinforcement of the odor-taste compound. Group CI developed a markedly greater aversion to the salt solution than did Group NCI (Figure 3). A repeated measures analysis of variance yielded a strong treatment effect ($F = 29.20$, $df = 1/14$, $p < .001$) as well as an extinction

Figure 3. Mean intake of sodium chloride solution in the presence of the odor of amyl acetate on the treatment day, and mean intake of sodium chloride solution in the absence of the odor of amyl acetate on the four subsequent test days.



effect across trials ($F = 7.60$, $df = 3/42$, $p < .001$). It is apparent that the animals in Group CI attributed their illness to the taste to a considerable greater degree than did those in Group NCI. It is remarkable that this effect was so strong in spite of the inhibitory training received by Group NCI as a result of Test Phase I.

These results served not only to substantiate the hypothesis that an odor can acquire active inhibitory properties, but, on a larger scale, they served also to support the Rescorla-Wagner model. Of particular importance is the fact that, although this model was originally devised to explain data which had been acquired via more traditional techniques, its implications are clearly generalizable to flavor aversion situations. This provides strong evidence for the general process theory of learning espoused by Revusky (1971).

Test Phase III

Once a stimulus has become a conditioned inhibitor of a given response, it is more difficult to transform that stimulus into an elicitor of that response than if the stimulus were either novel or latently inhibited. The demonstration of this effect comprises the retardation test of conditioned inhibition. In the present test phase, the odor of amyl acetate was presented together with unflavored water during repeated trials, and each time this was followed by toxicosis. It was expected that an aversion to the odor would develop more slowly in the group in which the odor was a conditioned inhibitor (Group CI) than in the group which had been given considerable prior experience with the odor but had not been given the initial conditioned inhibition training (Group NCI).

Method

Subjects. The same animals were used as in Test Phase II, and these retained their group designations.

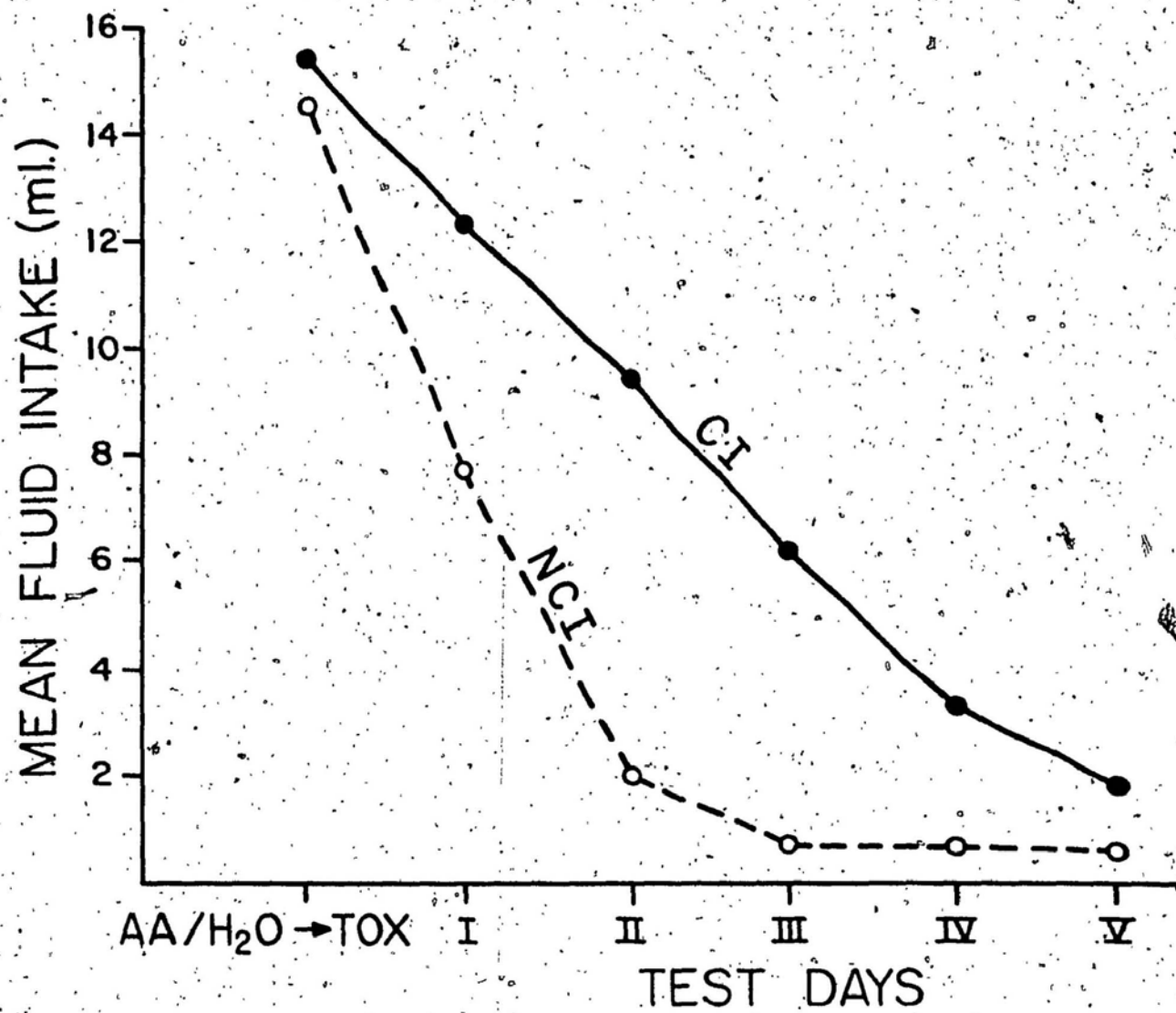
Procedure. Two water-only days were allowed after the last test day of Test Phase II (Day 51). On Day 54, unflavored tap water was presented to the rats for 10 min, together with the odor of amyl acetate. This was followed, for all animals, by the injection of 1% body weight of a 0.15 molar lithium chloride solution. Two recovery days were allowed on which water only was presented for 10 min in the absence of the odor. This procedure was repeated on five subsequent occasions, with odor-toxicosis pairings occurring on Days 57, 60, 63, 66, and 69. Each of these five treatment

sessions served as a test session as well; amount consumed was measured in order to determine the effects of the previous treatment, and toxicosis was again induced to further increase the aversion. Treatments were continued until most of the rats in Group NCI would no longer even taste the water.

Results and Discussion

Figure 4 shows the development of an aversion to the odor of amyl acetate for both groups as indicated by the reduction in water consumption. Although a virtually complete aversion was obtained for Group NCI after two odor-toxicosis pairings, the equivalent aversion never resulted in Group CI even after five such pairings; the difference in mean fluid intake between groups was still significant ($t = 1.93$, $df = 14$, $p < .05$, one-tailed) on the final test day (Day 69). A repeated measures analysis of variance across all five test days revealed a significant treatment effect ($F = 28.48$, $df = 1/14$, $p < .001$) as well as a significant acquisition effect across trials ($F = 49.16$, $df = 4/56$, $p < .001$). Thus, the retardation test provided the final piece of evidence that the odor had indeed acted as a conditioned inhibitor.

Figure 4. Mean intake of unflavored water in the presence of the odor of amyl acetate. Each test day represents a treatment day as well, with toxicosis always following the presentation of the odor and the water. The curves thus reflect the rate of acquisition of the aversion to the odor.



GENERAL DISCUSSION

Two recent edited volumes (Hinde & Stevenson-Hinde, 1973; Seligman & Hager, 1972) are sown with claims that construction of general theories of learning is futile at this time because of the existence of innate constraints on learning. Revusky's (1971) contrary interpretation is that, when innate constraints on learning are taken into account, general laws of learning are quite apparent. The present results support Revusky's position by showing that principles developed by Rescorla & Wagner (1972) for the explanation of compound classical conditioning and multiple-cue instrumental learning also hold for feeding behavior. Surprisingly, although Revusky's position and the Rescorla-Wagner model were developed independently and to account for apparently disparate phenomena, both are elaborations of the same basic principle: that there is associative interference in learning when two cues precede the same consequence. This convergence upon a common point from separate directions indicates that general theories of learning are more viable than is often believed.

There is a tendency to consider general learning theories to be convoluted explanations of behaviors which are more readily and naturally explicable as evolutionary adaptations to constant environmental contingencies. However, the present results are not predictable on an evolutionary basis because there is no obvious evolutionary reason to expect such a marked difference between latent inhibition and conditioned inhibition in the feeding

situation. Even if an evolutionary explanation were somehow to be conjured up, a second ex post facto explanation would still be needed for the results of Test Phase II, which seems to make sense only in terms of the Rescorla-Wagner model. Lavin's (1973) demonstrations that sensory preconditioning in flavor aversion learning develops in almost the same way as in traditional learning experiments illustrate the same point. If anything, sensory preconditioning of flavors ought to be evolutionarily maladaptive, because exposure to a flavor during a sensory preconditioning phase without contingent toxicosis ought to indicate to the animal that the flavor is harmless. Thus it is evident that general laws of learning cut across a wide variety of learning situations even when they do not result in specifically adaptive behavior.

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